Modern Concepts of Cardiovascular Disease

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HEART DISEASE AND PREGNANCY

Part I

Seven years have passed since the publication of the last comprehensive treatise on the relationship between the cardiovascular system and childbearing (Hamilton and Thomson, 1941). Since then, Chesley and his group have substantially enlarged our knowledge of the effect of pregnancy on the blood pressure. A controversy has been raging on the relative importance of the factors involved in congestive heart failure. Much of this discussion has an indirect, but important, bearing on heart disease and pregnancy. The treatment of infections, including subacute bacterial endocarditis, has been much advanced. Important contributions have been made to the diagnosis and treatment of congenital heart disease. Modern electrocardiography has rendered obsolete most previous work on electrocardiographic changes in pregnancy.

Many, relatively simple, issues remain undetermined; for example, changes in the size and rate of the heart and in blood pressure during pregnancy. The variations of these functions are so great both in the pregnant and the non-pregnant state as compared with the difference between the two states that statistically valid observations have not been obtained. Many of them seem to be facultative. They vary greatly from case to case or occur only in some pregnancies.

Considerable errors inhere in many determinations including apparently simple ones such as determinations of blood pressure or the diagnosis of valvular heart disease. There is an ever present tendency to draw general conclusions from a few, perhaps impressive, cases. In this field, it has been necessary to test many old concepts which were insufficiently supported, as well as to establish new facts.

The Demands of Pregnancy on the Circulation

Pregnancy increases the load on the circulation. The increase in weight calls for greater cardiac work. Not only is there more tissue to supply but locomotion requires more blood for the muscles. Though this demand exists beyond doubt, it cannot be measured accurately. It can, however, be greatly regulated through rest and exercise. The load of pregnancy is largely confined to the abdomen. It tends to shift the center of gravity of the body. To adapt itself to this shift, the body strains certain muscles, especially those of the trunk. In late pregnancy, the uterus displaces other organs and may render their function more difficult. If the patient is short and squat and the fetus large, the diaphragm and the organs which rest on it may be displaced. These changes are difficult to measure and show great individual variations. However, they do add to the work of the heart.

The variations in displacement of the diaphragm may explain the inconsistency of reports on vital capacity. Generally, vital capacity does not show marked changes during pregnancy. After delivery it suffers a sharp but temporary decline. A decrease of vital capacity during pregnancy may well be a sign of diminishing cardiac reserve.

The fetus has other effects on the circulation. Like other actively growing tissues, the pregnant uterus and the fetus require a relatively great blood supply. The caloric requirements may not exceed the normal capacity of the heart, but the mechanical effort of supplying a large additional vascular bed may be considerable. The blood vessels are numerous, large and tortuous. In the placenta, there are arteriovenous communications which Burwell has compared to arteriovenous fistulas. The amniotic fluid is relatively inert. These factors increase the load on the circulation. It has not been possible, however, to measure the extent of this increase.

Pregnancy affects the endocrine balance. Almost certainly, this change involves the circulation but it is as yet poorly understood. Both estrogenic and androgenic hormones are retained in the body. They may be responsible for the tendency to retain sodium and water. The changes in the pituitary and the thyroid glands and the adrenal cortex during pregnancy are barely more than suspected. They may well prove to have a bearing on fluid balance and the cardiovascular system. Plasma and total blood volume are increased during pregnancy and fall soon after delivery. This increase is a response to the greater demands for food and oxygen. It occurs in all pregnancies but varies greatly with the individual. It begins early in pregnancy and reaches its maximum of about 45 per cent above normal one month before delivery. Then the plasma and total blood volumes decrease to about 30 per cent above normal at term. During the puerperium, they recede to the nonpregnant normal. The increase is greater than can be accounted for by increase in body mass and the increase in plasma volume is proportionately greater than the increase in total blood volume. The reason for this disproportionate response is unknown. To suggest that it has to do with changes in the endocrine system simply invites another question concerning the mechanism of the endocrine changes. Hydremia of the blood is accompanied by hydremia of all the tissues. This fact is important in the development of congestive heart failure during pregnancy.

Consumption of oxygen is increased during pregnancy. The increase is to about 15 to 25 per cent above normal but varies greatly with the individual. It can be explained by increase in body tissues. During effort, utilization of oxygen is less efficient

than during rest. This may be, either because the tissues metabolize less efficiently or because the blood velocity is increased. However, it has not yet been proved conclusively that thyroid activity is increased during pregnancy or that the velocity of the blood is increased.

Ceteris paribus, increase in diastolic pressure results in an even greater increase in pulse pressure. Thus, increase in peripheral resistance requires the heart to expend increasingly greater energy to propel the same amount of blood. When the cardiac output is increased, this mechanism acts even further to the disadvantage of the heart. In normal pregnancy, this consideration is probably not important but when the blood pressure is increased because of peripheral vasoconstriction, the demands on the heart are greatly increased.

The viscosity of the blood and hematrocrit reading are increased in pregnancy. This renders the blood more difficult to propel, but these are minor factors.

The normal changes in blood pressure in pregnancy have been the subject of much discussion. Some observations indicate that arterioles and capillaries tend to contract more readily; on the other hand, observations that the blood velocity is increased would point to decreased peripheral resistance. Correspondingly, opinions are divided as to whether the blood pressure is increased or decreased in normal pregnancy. Actual observations are not conclusive and the issue is beclouded by the custom of considering all increase in blood pressure, beyond certain arbitrary limits, pathologic by the very fact of the increase.

The Mechanism Whereby the Heart Meets the Greater Demands

An increased volume of circulating blood means greater cardiac output. This is the principal factor in the increase in cardiac work. Increase in cardiac output is demonstrable from the twelfth week of pregnancy. It increases slowly to about the twenty-fourth week, then rapidly to the thirty-sixth week, when it reaches its maximum. From then on it decreases somewhat till term and, more rapidly, after delivery. The normal, nonpregnant level is reached after the first week of the puerperium. The maximal increase is 40 to 50 per cent of the nonpregnant output. This is more than the increase in oxygen consumption which, therefore, is less efficient.

Increased cardiac output can be achieved by increase in heart rate, by increase in stroke volume or by any combination of the two. The demonstrated increase in stroke volume is less than the increase in minute volume which indicates that there is also an increase in heart rate. The extent of this is difficult to prove directly because of the lability of the heart rate, which is especially marked in pregnancy.

Effect of Pregnancy on the Heart

During pregnancy, the work of the heart is increased because it must handle a greater blood volume. It has not yet been determined whether the normal heart meets this increased demand within its inherent reserve or whether it must hypertrophy to do so. A young healthy heart has such great reserve power that it might, *a priori*, be expected to assume this extra load without the necessity of hypertrophy, and the normal variations in cardiac weight are so great that they might well conceal a small degree of hypertrophy. By actual weight, it has not been possible to prove hypertrophy, neither on human hearts (which are difficult to obtain), nor by animal experiment. Roentgenologic measurements have not settled the matter, for such enlargement of the heart shadow as has been demonstrated might well have been produced by dilatation to accommodate greater stroke volume.

In many, if not all, women pregnancy encroaches on cardiac reserve. The amount of encroachment cannot be accurately expressed until some method applicable to pregnant women is devised of measuring cardiac reserve. This is a crucial problem in obstetrical cardiology, for sometimes, in apparently normal women, the reserve becomes depleted and the various manifestations of congestive heart failure develop.

Pregnancy is associated with a number of other cardiologic findings of less importance.

Depending upon the build of the patient and the size of the uterus, the heart may be displaced transversely. This change is apparent on roentgenographic examination. It is responsible for electrocardiographic changes, especially the development of a Q wave in lead III. It may be expected that this matter will be more accurately studied with unipolar leads.

The matter of functional murmurs is very difficult, for they vary normally so much that they do not readily lend themselves to statistical analysis. Many of the so-called murmurs of pregnancy are undoubtedly ordinary functional murmurs discovered during pregnancy. Some, however, do seem to be associated with the pregnant state. The mechanism of their production remains obscure in spite of many attempts to explain them. They are probably unimportant except in differentiation from "organic" murmurs.

Pregnancy, Delivery and Puerperium

Pregnancy begins to affect the cardiovascular system about the twelfth week. The demands then slowly increase for the next twelve weeks. Then the rapid growth of the fetus becomes associated with a more rapid increase in the cardiovascular changes. These reach a maximum at about thirty-six weeks and then slowly decrease until the time of delivery. No adequate explanation has been given for this pre-delivery decrease but it is an important integral part of pregnancy. It has practical therapeutic significance. The period about the thirty-sixth week is generally not the time to interrupt pregnancy for heart disease. The heart is then under maximal strain and will shortly be more favorably situated.

Much less is known about the demands made upon the heart by labor. During the pains, both the systolic and diastolic arterial pressures increase greatly, as does the venous pressure. The arterial pressure returns to normal between pains. The pulse rate increases during pains. The increase in total caloric requirements during labor is not very great and, somehow, the cardiovascular system appears to adjust itself to the strain, for it is rare for heart failure to appear in labor if pregnancy has been borne well. This observation should weigh heavily in decisions on the method of delivery of cardiac patients.

The effect of caesarean section on the cardiovascular system is even more obscure. No demonstrable "extra work" is performed by the heart during the laparotomy. It was, therefore, once thought that operation was less strenuous than natural delivery when cardiac reserve was narrow. So far, it has been impossible to compare the two forms of delivery by any form of physical measurement. It is also impossible to obtain exactly comparable groups of patients for statistical comparison. However, caesarean section has often proved an unsatisfactory manner of delivering a patient with narrow cardiac reserve and is being performed with increasing re-

luctance. Post-operative collapse is a frequent complication. A rational choice between natural delivery and caesarean section can be made, in the cardiac patient, only when the effects of major surgery on the cardiovascular system are fully understood. Following delivery, the various changes of pregnancy gradually return to normal. This is an irregular process and some changes persist for weeks or months, indicating that probably more than a simple mechanical adjustment is involved.

Formation of Abnormal Cardiac Impulses in Pregnancy

Cardiac arrhythmias present problems of etiologic relationship to pregnancy and of the effect they may have upon it.

Idiopathic auricular paroxysmal tachycardia presents great uncertainty. Any suggestion of an etiologic relationship to pregnancy is based on speculation and occasional observation rather than on sound and adequate study. Rarely does it endanger pregnancy. A great many paroxysms are short and stop spontaneously. Relatively few require treatment other than reassurance of the patient. Severe attacks may be treated with digitalis or, if more resistant, with an emetic (ipecac). Quinidine is said to be even more oxytocic than quinine, but opinions vary on the advisability of its use for arrhythmias during pregnancy. Personally, I avoid its use for pregnant women.

Extrasystoles, not associated with heart disease, are no more important in pregnant women than in others. A direct relationship to pregnancy has not been proved. In rare cases, they become so annoying that they require treatment. Digitalis is then the drug of choice but is often ineffective. In most cases, reassurance and, if the patient is nervous, sedation are preferable.

Auriculoventricular block is a matter of concern in pregnancy, especially when it is complete. It is rare. When it occurs as an isolated finding in a young woman, it is probably congenital. Enough cases have now been studied to establish the fact that in itself this lesion does not interfere with childbearing. When it occurs as part of other heart diseases, the outlook is determined by the cause. If the patient develops Adams-Stokes attacks, the outlook is changed for the worse.

Except in rare cases, bundle branch block is evidence of serious heart disease and experience with it has been unfavorable. It should be evaluated with reference to the underlying heart condition.

The Effect of Pregnancy on the Myocardium

For many years fragmentary evidence has appeared that pregnancy might affect the myocardium. It has been asserted that too much childbearing might wear out the heart. Occasionally, fatal heart failure may appear in the puerperium, and the myocardium has then been found to be the site of nonspecific degenerative changes. Changes in the T waves have been found in the electrocardiogram in some cases. Some pregnant women complain of symptoms of cardiac incompetency: shortness of breath on effort; orthopnea and edema. They may have advanced congestive heart failure and yet recover after delivery so that they then show no evidence of organic heart disease. These findings are uncommon and have not yet been co-ordinated into established clinical entities.

Organic Heart Disease

General considerations — Childbearing increases the work of the heart. With rare exceptions, the normal heart has ample reserve to carry this increase. In organic heart disease, the outlook depends upon the available reserve, and the factors which generally influence prognosis apply with the added consideration of increasing cardiac strain. In some pregnant women with heart disease heart failure develops as pregnancy advances, but if they are capable of carrying the pregnancy to the thirty-sixth week, their hearts rarely fail near term, during labor or in the puerperium. Frequently, failure can be controlled if it is discovered early and treated well. Early diagnosis of the failure is, therefore, essential. Pregnant women who have cardiac disease should be examined frequently for early signs of cardiac failure. This procedure, together with a better understanding of the problem, has reduced the mortality rate from the complication to about 2 per cent.

The prognosis is influenced by some factors which are common to the various forms of heart disease. It is definitely better for patients who are less than 30 years of age and becomes more serious with advancing age. Cardiac enlargement adds to its gravity; when a very large heart is present the prognosis is poor. When auricular fibrillation is a sign of advanced heart disease, it increases the chances of death about ten times.

In spite of many clinical impressions and of the statement made in the section on the "Effect of Pregnancy on the Myocardium," it has not been possible to prove that repeated childbearing in itself has aggravated organic heart disease, and there is no hypothetical reason why it should. In so far as the effects of pregnancy are known, they affect only *cardiac reserve* temporarily during childbearing. There is no accepted evidence that pregnancy affects the essential disease processes of the heart or that its influence extends much beyond the period it is effective.

The physical environment, especially domestic and economic conditions, may greatly influence the outlook. They require the intelligent co-operation of the patient, her family and often social agencies.

Until recently, subacute bacterial endocarditis was a disastrous complication. Fortunately, it is rare. It can now be treated effectively in pregnancy, for there is no evidence that antibiotics endanger gestation.

Specific Forms of Heart Disease

Congenital heart disease—Cardiac diseases of congenital type are gaining in relative importance. Differential diagnosis has improved and surgical operations in some cases can repair or compensate for the congenital defects. While congenital heart diseases are relatively rare among pregnant women, the aggregate number is considerable and gradually clinical pictures are evolving, each requiring specific considerations.

As a group, pregnant women who have congenital cardiac disease run the risk, common to most forms of heart disease, of congestive failure and of subacute bacterial endocarditis, but some of them present the additional danger of reversal of the blood stream immediately following labor or caesarean section which has proved fatal in a number of cases.

Cyanosis is an unfavorable feature in proportion to its degree and persistence. When marked, it constitutes a contraindication to pregnancy. Congenital defects of the heart, which increase its work, may lead to congestive heart failure. Their course is associated with hypertrophy of those chambers which are under strain. Consequently, the encroach-

ments upon cardiac reserve and hypertrophy of individual chambers indicate the severity of the disease.

Several congenital lesions are now so well identified in relation to pregnancy that specific statements can be made concerning them. Women with auricular septal defects can generally accomplish childbearing. When the defect is extreme, the strain upon the heart may lead to congestive failure. This is one lesion in which venous-arterial shunt is feared. Paradoxical embolism may occur. Frequently other lesions are associated; some patients with accompanying mitral stenosis have done well. Patent ductus arteriosus involves three risks: congestive heart failure, subacute bacterial endocarditis and venous-arterial shunt. Congestive failure is the natural termination for patients with this lesion. If it is approaching, pregnancy may, and often does, precipitate it. If the cardiac reserve is ample, the outlook is good because the other two risks rarely materialize. However, accompanying lesions frequently occur and modify the prognosis. Isolated septal defects generally present a favorable prognosis, depending somewhat on the size of the defect. The diagnosis must be made with particular care, for other defects, especially pulmonary stenosis, are frequently present and modify the outlook. Septal defects are peculiarly susceptible to bacterial endocarditis. Some patients have collapsed with venous-arterial shunts following delivery. Congestive failure is an additional, though rare, risk. Pulmonary stenosis, frequently associated with ventricular septum and other defects, is often associated with cyanosis and might be expected to be a serious threat to pregnancy. As a matter of fact, pulmonary stenosis is rarely diagnosed in pregnant women, but in all of the cases which I have seen reported the patients have survived, most of them without much difficulty. They should be treated conservatively but observed closely. Coarctation of the aorta and pregnancy has now been reported in many cases; in most cases normal delivery is accomplished though there is a definite risk that the complications to which these patients are specially liable, vascular accidents and congestive heart failure, may be precipitated by the pregnancy. They should not undertake pregnancy lightly, especially not past the age of 30 years.

Other congenital defects are of little interest to the obstetrician: they are either insignificant, undiagnosed or incompatible with conception. It is yet too early to express an opinion on the effect which modern surgical treatment will have on childbearing in patients with congenital heart disease.

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ANNUAL MEETING

The Annual Meeting and Twenty-Second Scientific Session of the American Heart Association will be held in Atlantic City, New Jersey, on June 3 and 4, 1949. The Chalfonte-Haddon Hall will be the headquarters for all meetings and for the Annual Dinner which will take place on Saturday evening, June 4.

The Chairman of the Program Committee for the Annual Scientific Session is Dr. Eugene A. Stead, Jr., Duke University School of Medicine, Durham, North Carolina. All who desire to present papers at the meetings in Atlantic City on June 3 and 4 should forward to Dr. Stead an abstract (in triplicate) of the proposed presentation of 300-words. The deadline for the receipt of abstracts is March 1, 1949.

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